Treatment of Facial Palsy –A Review

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Abstract

A common presentation of facial palsy in childhood is acute lower motor neuron palsy. An etiological agent is not identified in a number of cases and the condition spontaneously regresses. A few cases are caused by a variety of pathologies—many of which may have significant co-morbidity and mortality associated. This article presents a review of facial palsy and its treatment.

Key words: Facial palsy, Facial Nerve, Treatment

Introduction

Introduction to the anatomy

The facial nerve (VII) exits from the pons at the pontomedullary junction, and gets into the skull through the way of internal auditory meatus, and travels along the facial canal. The nerve travels in very close proximity to the medial wall of the inner ear as well as the mastoid cavity. When it travels through the petrous temporal bone, branches exits to supply the lacrimal glands, the stapedius muscle of the inner ear, sensation for the skin of auricle, the sublingual and submandibular salivary glands, and anterior two thirds of the tongue as taste fibres. The facial nerve leaves the skull via the stylomastoid foramen and terminal branches tends to supply the muscles of facial expression.

Applied aspects for Facial palsy

Bilateral cortical innervation of the muscles of the upper face (orbicularis oculi and frontalis) signifies that complete facial paralysis is the classical feature of with lower motor neurone lesions. The extent of additional impairment depends on the site of injury. Proximal lesions are related with impaired lacrimation, hyperacusis, and loss of taste on the anterior two thirds of the tongue.

The pathophysiology of idiopathic facial nerve palsy is not known. Viral illness for around 2-4 weeks leads to such a condition. Nerve or immune demyelination may be responsible for the underlying neuropathy which is due to active viral invasion. physical swelling of the nerve plays a significant pathological role, is still unkonwn. A variety of specific pathologies can also produce an acute lower motor neurone facial paralysis. **Table 1** lists some important examples. Acute facial paralysis may manifest itself as a presenting symptom of one such specific disease, or, as is more common, occur as a complication during the course of a recognized illness.

Bells palsy

Facial palsy of rapid onset related to a lesion of the nerve within the facial canal is known as Bells palsy.[1] While the majority of authors use the eponym to describe idiopathic facial palsy alone, this is not universal. Descriptive terms are less open to confusion and tend to focus the mind on potential differential diagnoses.

There is controversy related to the appropriate investigation of children presenting with acute facial paralysis. A thorough clinical history and a detailed physical examination is required to proceed . An urgent specialist referral is required for the children with atypical signs or symptoms (see **table 2**). in patients presenting with facial paralysis, otoscopy is mandatory. Where adequate auditory acuity cannot be confirmed an audiogram should be arranged. A well-recognized and a rare cause of facial paralysis is hypertension; all children must have their blood pressure checked.

The utility of further investigation, in the absence of additional symptoms or specific findings on physical examination, is debatable.

If additional neurological abnormalities are identified or if malignancy is suspected, radiological imaging is suggested. Magnetic resonance imaging is especially helpful in identifying brainstem pathology. Intratemporal portion of the nerve can be evaluated using high resolution computed tomography scanning. sections of affected nerve in idiopathic facial palsy could be detected using.[4] contrast enhanced magnetic resonance imaging but the major disadvantage of this investigation is that its cost and being invasive.

In leukaemic recurrence cases, facial paralysis is common in both adults and children. A small number of cases of facial paralysis in children, occurring as a presenting feature of leukaemia, have also been described. As a result, in all children presenting with in acute lower motor neurone facial paralysis, routine performance of a full blood count have been suggested by some authorities[5] . Six reported cases have been reported of leukaemia presenting with facial paralysis in children by us.[6–11] In all but one case,[7] additional symptoms, documented at presentation, would prompt further investigation. In four of the reports haematological findings are documented.[8–11] Blood count abnormalities were present in each case, changes can be subtle and initial haematological findings were discounted or overlooked in three of the four cases.

Prognostic information can be obtained using neurophysiological studies and are useful in evaluating lesions that have not resolved at one month. While several tests exist, many require significant cooperation on behalf of the subject. Two of the simpler tests are the measurement of fibrillation potentials (as part of electromyography) and recording of the blink reflex. Fibrillation potentials in muscles which have lost their innervation are detectable, after approximately three weeks. Significant axonal degeneration is depicted on its presence. Blink reflex tests are utilized.

Table 1 Acquired facial nerve paralysis can be due to causes listed below

• **Infective or inflammatory**
• **Otitis media**
• **Mastoiditis**
• **Herpes zoster (Ramsay Hunt syndrome[19])**
• **Temporal lobe abscess**
• **Varicella**
• **Mumps**
• **Meningitis**
• **Encephalitis**
• **Mycoplasma[16]**
• **Cat scratch disease[26]**
• **Kawasaki disease**
• **Guillain–Barre syndrome HIV[27]**
• **Lyme disease**
• **Trauma**
• **Facial burn[28] Basal skull fracture Blunt and penetrating trauma Facial surgery**
• **Neoplastic**
• **Leukaemia Cerebellar astrocytoma Rhabdomyosarcoma**
• **Haematological**
• **Haemophilia[29] Histiocytosis**
• **Congenital**
• **Melkersson–Rosenthal syndrome Osteopetrosis[28] Intracerebral arteriovenous malformation[28]**
• **Other**
• **Hypertension**

Table 2 Symptoms and signs indicative of possible additional pathology where urgent specialist referral is required

• Ear ache Hearing loss Pain or paraesthesia
• Any abnormality on otoscopy—including otitis media
• Associated cranial neuropathies or other neurological signs
• Hypertension
• Lymphadenopathy,
• pallor or bruising Vesicles in external meatus or on soft palate
• Single branch involvement Gradual progression of paralysis beyond 3 weeks
• Recurrent Mastoid swelling

An early ipsilateral facial (VII) motor response is produced, followed by a bilateral late response by Unilateral stimuli, transmitted via the trigeminal nerve (V),. to elicit the reflex a variety of stimuli can be used, for example, electrical stimulation of the supraorbital nerve or a puff of air directed onto the cornea. Electrodes placed over both infraorbital ridges give the readings. Assess to facial innervation can be obtained by evaluating Absence or delay in the late response. Neither test can provide useful information in the acute setting, nor can they differentiate the pathological processes responsible. None the less, such tests can provide objective assessment of facial nerve function and reassure parents and children.

The treatment of idiopathic facial palsy is also controversial. Duration of paralysis and the risk of long term impairment can be reduced by the use of steroids early in the course of the disease. A meta-analysis of randomised control trials, performed in adults, seems to support this view.[12] A larger portion of patients treated with steroids recovered completely and mean time to recovery was shorter. Treatment appears to be more effective when started within 24 hours of onset.[13] The benefits of steroid treatment have yet to be proven in children.[14] As the vast majority of children will recover completely, with or without treatment, a very large intervention study would be required to show any significant effect.

In the pathogenesis of idiopathic facial palsy relates to Herpes simplex and varicella zoster viruses have, [15 16] and the routine use of antiviral agents has been suggested. acyclovir alone is less efficacious than steroid treatment has been shown in studies.[17] The use of acyclovir in combination with steroids does not improve the outcome of idiopathic facial palsy.[18]

Reactivation of the varicella zoster virus in the geniculate ganglion causes Ramsay Hunt syndrome (or herpes zoster oticus). Unilateral facial paralysis is accompanied by herpetic vesicles in the external auditory canal or on the soft palate. The prognosis for this condition is not as good as that of idiopathic facial palsy.[19] Aggressive treatment with intravenous acyclovir, possibly in combination with steroids, is recommended.

in adults, A number of other treatments, aimed at improving outcome in idiopathic facial palsy, have been reported.

Summary

A thorough physical examination is required in Patients presenting with acute lower motor neurone facial paralysis. Full neurological examination, otoscopy, and blood pressure measurement are mandatory. Further investigation is unnecessary, in the absence of any abnormal symptoms or signs. No clear evidence exists that any form of treatment improves outcome of idiopathic facial palsy in children till date. full recovery is seen in 95 % of children in function,[24 25] most within the first three weeks of the illness. Protection of the cornea, with artificial tears and overnight patching, is normally all that is required. Follow up is advisable. Neurophysiological assessment is helpful in patients with weakness persisting beyond three weeks. Patients treated with vitamin B and steroids recovered faster than those treated with steroids alone. In a randomised, blind study, patients treated with 100% hyperbaric oxygen recovered faster than those treated with steroids and 7% oxygen at the same pressure. Group receiving the higher oxygen concentration showed better outcome.[21]

Surgical decompression of the facial nerve canal is no longer considered an effective, or appropriate, as treatment for patients with idiopathic facial palsy.

Persistent facial weakness has considerable functional and cosmetic implications. Significant psychosocial morbidity is caused due to disfigurement. The small numbers of children falling into this group require expert assessment. Feedback training and exercise programmes have been shown to provide some benefit in adult patients with long standing facial nerve paralysis.[22] Surgical techniques, aimed at improving function and cosmetics, include nerve repair, graft, or transposition. Attachment of the distal end of the affected facial nerve to another afferent cranial nerve trunk, for example, the contralateral facial nerve, or a hypoglossal "jump graft" was a part of Nerve transposition. This technique must be undertaken within two years of paralysis.

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